Clinical Investigation

Pericardial Tamponade and Large Pericardial Effusions

Causal Factors and Efficacy of Percutaneous Catheter Drainage in 50 Patients

Mehmet Kabukcu, MD
Fatih Demircioglu, MD
Ekrem Yanik, MD
Ibrahim Basarici, MD
Filiz Ersel, MD

In 50 patients treated from January 1998 through March 2002 for pericardial effusion and tamponade, we retrospectively investigated the efficacy of percutaneous placement of an indwelling pericardial catheter guided by 2-dimensional echocardiography and fluoroscopy. We also investigated causation.

In 80% of the patients, we were able to determine specific causes through clinical, serologic, and cytologic investigation: cancer in 15 patients, chronic renal failure in 11, systemic lupus erythematosus in 2, rheumatoid arthritis in 2, Dressler syndrome in 2, tuberculosis in 1, blunt chest trauma in 1, purulent pericarditis in 1, and probably viral pericarditis in 5. No specific cause could be determined in 10 patients (20%). We did not observe any complication due to the procedure. Two patients died during hospitalization. After hospitalization, 9 patients with metastatic cancer died within 3 months. A 2nd percutaneous drainage procedure was required in 2 cancer patients. Recurrence of pericardial effusion and tamponade and the requirement of pericardiectomy occurred in 2 patients with perfusion of unknown cause and in 1 patient with perfusion due to rheumatoid arthritis. Histologic examination of pericardial tissue in patients with idiopathic disease showed fibrinous pericarditis but no causal factor. In the group with idiopathic pericardial effusion, 2 patients with multiple mediastinal lymphadenopathy underwent mediastinal exploration; biopsy revealed nonspecific lymphadenitis and fibrinous pericarditis.

In patients with large pericardial effusions and tamponade, the specific cause was in most cases already known or obtained by initial clinical and laboratory investigation. Sufficient cardiac decompression was achieved by percutaneous pigtail catheter drainage. ***(Tex Heart Inst J 2004;31:398-403)***

Key words: *Arthritis, rheumatoid/complications; cardiac tamponade/therapy; catheters, indwelling; drainage/methods; echocardiography; lupus erythematosus, systemic/complications; neoplasms/complications; pericardial effusion/therapy; pericardial effusion/ultrasonography; pericardiocentesis/methods; pericarditis/complications; punctures/methods; uremia/complications*

From: *Department of Cardiology, Akdeniz University, School of Medicine, 07070 – Antalya, Turkey*

Address for reprints: *Dr. Mehmet Kabukcu, Akdeniz Universitesi, Tip Fakultesi, Kardiyoloji ABD, 07070 – Antalya, Turkey*

E-mail: kabukcu@akdeniz.edu.tr

Pericardial tamponade, a life-threatening condition caused by the accumulation of fluid in the pericardial sac, is treated by drainage.[1,2] Surgical placement of a subxiphoid tube is the preferred technique for draining a small amount of effusion in patients with quickly developing pericardial tamponade, such as those with acute traumatic hemopericardium.[3-5] For patients with massive effusion and slowly developing pericardial tamponade, there are 2 principal methods: percutaneous catheter drainage and surgical tube drainage.[5-9]

The infrequency of effusive and compressive pericardial disease limits the feasibility of large, randomized studies to compare the effectiveness of treatment strategies.[7] The choice of drainage method depends on the cause of the effusion, the patient's general health, the physician's experience and preference, and the facilities available. Because the management of cardiac tamponade is governed to such a large extent by institutional practice, it remains controversial.

The causes of pericardial effusion change over time. At the time that we undertook this review, no causal evaluation of pericardial tamponade had been performed recently in a developing country. Moreover, our ability to determine specific causes in patients who had no apparent underlying disease was unknown, without access to pericardial tissue biopsy specimens.

A major risk of percutaneous pericardiocentesis is laceration of the heart, coronary arteries, or lungs. If needle pericardiocentesis is performed at the bedside without echocardiographic guidance or hemodynamic monitoring, the risk of life-threatening complications is as high as 20%.[10] Echocardiograpic guidance increases the success rate of pericardiocentesis by reducing these complications.[3]

We report our experience with a series of 50 patients who presented with large pericardial effusions and tamponade and underwent placement of an indwelling catheter by echocardiographic and fluoroscopic guidance. We set out to answer the following questions: 1) Can clinical, serologic, and cytologic evaluation of patients determine the specific cause of tamponade? 2) What is the role of pericardial tissue biopsy in determining causation? 3) Is pericardial drainage with a catheter both safe and therapeutically sufficient?

Patients and Methods

This retrospective review was carried out at Akdeniz University Hospital. From January 1998 through March 2002, we performed percutaneous catheter drainage treatment in 50 patients who presented with massive pericardial effusions complicated by pericardial tamponade. Initial puncture of the pericardial cavity was performed under echocardiographic guidance. Placement of catheters by means of the Seldinger technique was controlled fluoroscopically in all procedures. There were 16 females and 34 males. The mean age was 51 ± 17 years (range, 12–80 years). Patient follow-up was performed for a median of 18 months (range, 3 months to 3 years).

The diagnosis of evident pericardial effusion was established echocardiographically by determining whether the echo-free space (representing pericardial fluid) surrounding the heart was more than 10 mm deep in front of the right ventricle and beyond the left ventricle (Fig. 1). Pericardial tamponade was diagnosed by observing compression of the right ventricle in diastole by echocardiography, in the presence of tachycardia (heart rate >100 beats per minute) or pulsus paradoxus (>10-mmHg decrease in systolic blood pressure on inspiration).[11]

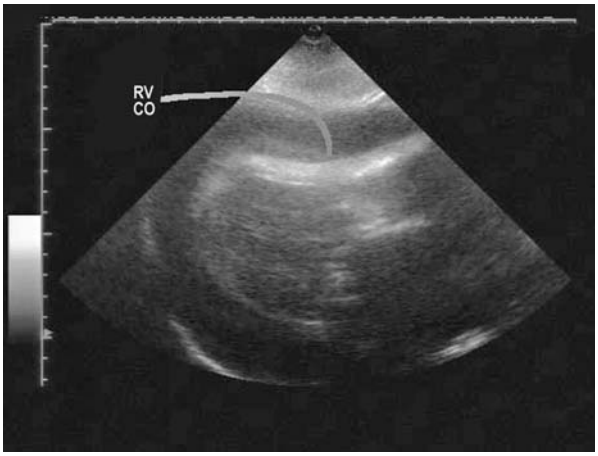

Fig. 1 *Subcostal echocardiographic image of pericardial tamponade: the echo-free space (representing pericardial fluid) surrounding the heart is more than 10 mm deep in front of the right ventricle and beyond the left ventricle. Note the compression of the right ventricle in diastole.*

RV CO = right ventricular collapse

We excluded patients with pericardial tamponade due to a small amount of effusion or to localized pericardial effusion. In addition, we excluded patients who developed pericardial tamponade after surgical intervention or cardiac catheterization procedures, and patients who developed tamponade due to mechanical complications of an acute myocardial infarction, such as rupture.

All patients were transported to the catheterization laboratory after diagnosis of pericardial tamponade. Patients were kept in a semi-sitting position by supporting their backs. The optimal puncture site and the direction for pericardiocentesis were determined by 2-dimensional (2-D) echocardiographic guidance. The transducer localized the pericardial fluid,[12] and pericardial puncture was performed under sterile conditions with an 18-gauge needle (continuously aspirated during the puncture). After the pericardial space was reached, a soft-tipped 0.038 guidewire (Terumo; Tokyo, Japan) was passed and the needle removed. The location of the wire was confirmed when a loop formed within the pericardium. A 7F Cordis introducer sheath (Johnson & Johnson; Miami, Fla) was placed in the pericardial space over the soft-tipped Terumo guidewire. A multiholed (end-and-side) soft 7F pigtail catheter was advanced into the pericardial sac, inside the introducer sheath and over the guidewire. The guidewire was then removed, and the pericardial fluid was suctioned from the catheter tip into a reservoir, through a closed drainage system. The catheter was left in the pericardial space for at least 72 hours. During suction, echocardiography was performed twice a day, and the amount of drainage was observed. We administered sulbactam plus ampicillin intravenously (1.5 g, 4 × per day) before the puncture and during drainage.

The catheter and sheath were removed when the pericardial fluid was completely or almost completely emptied as determined by echocardiography (the remaining fluid was less than 5 mm in depth both at the anterior and posterior sites) and, simultaneously, the drainage volume decreased to less than 100 cc per 24 hours.

Specimens of the fluid were sent for chemical and cytologic study, for culture, and for Ziehl-Neelsen staining. Thoracic and upper-abdominal computed tomographic and upper- and lower-abdominal echocardiographic examinations were performed in all patients.

Results

Procedural Success and In-Hospital Outcomes

We evaluated procedural results of the 50 patients. In all patients, pericardial puncture was performed via the subxiphoid region. Puncture was achieved on the 1st attempt in 40 patients, and 8 patients required a 2nd or 3rd attempt. Four or more attempts to puncture were needed in only 2 patients. No patient required surgical intervention because of a failed puncture. No early complication of pericardial puncture, such as cardiac perforation, ventricular fibrillation, or pneumothorax, was observed. There was no death as a consequence of the procedure.

The average volume of pericardial fluid drained was 1,450 ± 280 cc. For all patients, the amount of fluid drained on the 2nd and 3rd days was less than 100 cc and 50 cc, respectively. No fever, infection, or hematoma was observed during follow-up.

The macroscopic appearance of the drained materials was serous in 12 patients, hemorrhagic in 37 patients, and purulent in 1 patient. Biochemical analysis revealed that all specimens were exudates according to Light's criteria (pericardial–plasma protein ratio >0.5, pericardial–plasma LDH >0.6, or pericardial fluid LDH >200 U/L) The mean LDH (lactic dehydrogenase) level of the pericardial fluid was 1,952 ± 1,207 U/L, and the mean total protein level was 4.9 ± 1.3 g/dL; the ratio of pericardial protein to plasma protein was 0.71 ± 0.12.

Causal Evaluations

Frequent Causes of Effusions. Malignancy, uremia, and possible viral infections were the most common causes of large pericardial effusions and cardiac tamponade (Table I).

TABLE I. Underlying Causes or Condition of Pericardial Tamponade in 50 Patients

Cause	No. of Pts. (%)
Cancer	15 (30)
Uremia	11 (22)
Virus (probable)	5 (10)
Connective tissue diseases	4 (8)
Dressler syndrome	2 (4)
Tuberculosis	1 (2)
Purulent pericarditis	1 (2)
Trauma	1 (2)
Idiopathic disorder	10 (20)

Malignant pericardial effusion was found in 15 patients (30%): 11 patients (22%) had pericardial tamponade during the course of their known cancers. In the other 4 patients (8%), pericardial tamponade was the 1st manifestation of the disease, and causal investigations revealed the cancer.

Uremic pericardial effusion was found in 11 patients (22%); all of these patients had been in routine dialysis.

Viral pericarditis was the probable cause of pericardial effusions in 10% of all cases (5 patients). The diagnosis of viral pericarditis was assigned when the patient had a history of upper respiratory tract infection within 15 days before the onset of tamponade and when other causal factors could be excluded.

Infrequent Causes of Effusions. In 2 patients, pericardial effusions were due to systemic lupus erythematosus. Diagnoses were confirmed by anti-DNA positivity. The other 2 patients with pericardial effusions due to connective tissue disorders (Table I) had been diagnosed with rheumatoid arthritis before the onset of tamponade.

Tuberculosis was the cause of pericardial effusion in 1 patient. Ziehl-Neelsen staining of the pericardial fluid sample showed acid-resistant bacteria.

Dressler syndrome was detected in 2 patients. In the 1st of these patients, pericardial effusion occurred 2 months after an anterior myocardial infarction, and in the 2nd it occurred 2 months after an inferior myocardial infarction.

In 1 patient, the pericardial effusion was due to purulent pericarditis, and methicillin-sensitive *Staphylococcus aureus* was grown in the culture. The case was treated by intravenous sulbactam plus ampicillin (1.5 g, 4 × per day).

Pericardial effusion and tamponade due to blunt chest trauma was established in 1 patient. Ten days after the injury, he was transported from another hospital for progressive dyspnea. Because of concomitant adult respiratory distress syndrome (ARDS), a percutaneous drainage method was selected for this patient.

We could not identify the specific cause of pericardial effusion in 10 of the patients (20%).

Hospital Mortality

Two patients died in the hospital. The patient with pericardial tamponade that developed after blunt chest trauma died of ARDS on the 4th day after successful pericardiocentesis. Another patient died 7 days after pericardiocentesis because of multiple-system organ failure (ARDS, renal failure, and disseminated intravascular coagulopathy).

Results of Long-Term Follow-Up

Follow-up ranged from 3 months to 3 years (mean, 18 ± 9 months). During the follow-up period, symp-

tomatic recurrence of pericardial effusion occurred in 2 patients with idiopathic pericardial effusion and in 1 patient with rheumatoid arthritis. Surgical pericardiectomy was required in these patients. A 2nd pericardiocentesis procedure was required in 2 patients with malignant disease. Nine patients with pericardial tamponade due to malignant metastatic disease died within 3 months of presenting with tamponade.

Discussion

Causal Evaluation of Large Pericardial Effusions and Tamponade

In most of our patients, it was possible to identify an underlying condition or disease as the cause of the effusion. An apparent underlying illness (metastatic malignant disease, chronic renal failure, rheumatoid arthritis) was known in 24 patients (48%), at the time of their presentation with tamponade. In addition, in 11 patients (22%), clinical, serologic, cytologic, and radiologic investigation suggested the cause: newly diagnosed malignant disease, systemic lupus erythematosus, Dressler syndrome, chest trauma, tuberculosis, and purulent pericarditis). A recent history of respiratory infection together with signs of inflammation (exudative effusion), responsiveness to anti-inflammatory drug therapy, and no recurrence at follow-up led us to a diagnosis of probable viral pericarditis in 5 patients (10%). These findings suggest that initial assessment with clinical, serologic, and radiologic investigation, followed by careful monitoring, can enable the discovery of cause in most cases of pericardial tamponade. In our series. a causal diagnosis was obtained by means of clinical assessment in 80% of patients. As in our series, Sagristà-Sauleda and colleagues[13] reported that, in many patients, pericardial effusions were due to a known underlying disease or condition; in patients without known underlying diseases, signs of inflammation, the size of the effusion, and the presence or absence of cardiac tamponade can be helpful in establishing causation. The origins of our patients' pericardial infusion were similar to those found by Colombo's group.[14] In their series, the most frequently encountered causal factors were neoplastic diseases (36%), idiopathic pericarditis (32%), and uremic pericarditis (20%).

Idiopathic Pericardial Effusions and the Requirement of Tissue Biopsy

In cases in which the causal diagnosis of pericardial effusion cannot be established by radiodiagnostic and biochemical means, there remains considerable difficulty in diagnosis. These effusions are said to be idiopathic, and 10 patients (20%) in our series had idiopathic pericardial effusions. During follow-up, 5 of these patients had no recurrence. These patients are still asymptomatic and under close observation. One patient with idiopathic pericardial effusion died during hospitalization as a consequence of multiple-system organ failure.

Two of the patients with idiopathic pericardial effusion underwent pericardiectomy for recurrence of effusion. Pathologic examination of pericardial material revealed fibrinous pericarditis, which described a condition but did not yield a specific causal diagnosis. In another 2 patients—whom we determined by thoracic computed tomography to have multiple mediastinal lymphadenopathy—surgical intervention was performed to obtain mediastinal and pericardial tissue biopsy specimens. Pathologic examination revealed nonspecific lymphadenitis and fibrinous pericarditis. In all 4 patients (8%) who were evaluated by surgical procedures, the interventions could not contribute to establish causal diagnosis. These findings suggest that pericardial tissue biopsy is not essential during the acute tamponade phase and should be performed only if effusion recurs and if the specific cause cannot be determined by clinical and laboratory investigations.

Reducing the Risk of Pericardiocentesis

Percutaneous pericardiocentesis carries the risk of cardiac and coronary laceration, pneumothorax, liver trauma, and death.[5] The efficacy and safety of echocardiographically guided puncture and catheter-based percutaneous drainage of the pericardium was confirmed again with this series. Our complication rate will become more realistic as our number of cases increases. In the meanwhile, we have similar reports from other investigators, to the effect that their complication rates for echocardiographically guided percutaneous pericardiocentesis fall within an acceptable range.[15-17]

Hospital Mortality

The fact is that early decompression must be obtained in patients with cardiac tamponade, or their long-term prognosis is worsened.[18] In our series, 2 patients died. One of these deaths was due to cardiac tamponade after blunt chest trauma; the patient was referred to our hospital 7 days after the injury. Surgical exploration is the 1st choice in the diagnostic evaluation and treatment of traumatic pericardial effusions. However, in this case, we performed catheter-based pericardiocentesis because of the concomitant ARDS. Despite successful cardiac decompression, the patient died of ARDS 4 days after the procedure. The other patient who died was in group with idiopathic pericardial effusion. This patient had been evaluated and treated in another hospital for progressive dyspnea and was referred to our hospital because of dyspnea, renal failure, and disseminated intravascular coagu-

lopathy. This patient died due to multiple-system organ failure, although pericardiocentesis had been successful. These 2 cases suggest, as did the report of Larose and associates,[18] that long delays in treatment are not suitable for patients with pericardial tamponade.

Specific Causation

In all 11 patients with known malignant pericardial effusions, we found widespread pulmonary, pleural, and mediastinal metastasis. Furthermore, in 2 patients in this group, pericardial effusions formed while the patients were receiving mediastinal radiotherapy. In 2 patients with malignant pericardial effusions, initial drainage achieved clinical relief, but recurrent pericardial effusions and tamponade developed during follow-up, so a 2nd pericardiocentesis was required. Our 9 patients who did not receive mediastinal radiotherapy before development of cardiac tamponade were all dead within 3 months of the intervention. In addition, 2 cancer patients who experienced pericardial tamponade during mediastinal radiotherapy died 3 and 6 months after their procedures. This high mortality rate among our cancer patients with pericardial effusion is primarily related to the poor prognosis of malignant disease.[19] Because of the poor long-term prospect of survival in these patients and their physical debilitation as a consequence both of the primary disease and of ongoing chemotherapy and radiotherapy, we believe that pigtail catheter drainage is preferable to surgical intervention.

The prevalence of malignant disorders in patients whose 1st clinical manifestation is pericardial tamponade is unknown.[20] In our 4 patients whose 1st clinical manifestation upon hospitalization was cardiac tamponade, we diagnosed the malignancy after pericardiocentesis. In 1 of these patients, we determined the histocytologic findings (from examination of the pericardial fluid) to be consistent with adenocarcinoma. In this patient, biopsy of a right supraclavicular lymph node specimen had confirmed the adenocarcinoma diagnosis, but no primary focus could be defined. The patient was treated with chemotherapy and mediastinal radiotherapy. Balghith and coworkers reported a similar case.[21] In another patient, we established a diagnosis of ovarian carcinoma and peritoneal carcinomatosis; she was treated by surgery and adjuvant chemotherapy. The 3rd patient was diagnosed with a metastatic malignant epithelial tumor of unknown origin and was treated with systemic chemotherapy. In the last patient, we found diffuse metastatic malignant disease with bronchopleural fistula and empyema; he died 14 days after the drainage procedure.

Uremic pericardial effusion is an expected complication in the course of chronic renal failure. In our series, 11 patients with known chronic renal failure presented with overt hypotension and tachycardia during their dialysis sessions, which suggested pericardial tamponade. All these patients had successful pericardiocentesis and drainage, which yielded hemorrhagic pericardial fluid attributable to a coagulopathic state of uremic disease. In follow-up, the frequency of dialysis sessions was increased, and no patient developed recurrent effusion. We consider percutaneous catheter drainage to be an effective treatment for uremic pericardial effusions and tamponade.

In connective tissue diseases, clinically insignificant pericardial effusion is common but pericardial tamponade is rare.[22-27] We established the diagnosis of systemic lupus erythematosus in 2 patients whose 1st presenting symptom was cardiac tamponade. Some cases in which cardiac tamponade was the 1st presenting manifestation of the disease have been reported in the literature.[22,24] Pericardial tamponade developed in 2 of our patients while they were in follow-up for rheumatoid arthritis. Both of these patients were treated by increasing their dosage of systemic corticosteroids and intrapericardial steroids (by injection), after successful pericardial drainage. However, 1 of the 2 patients had a recurrent massive pericardial effusion after 2 months and required a 2nd pericardiocentesis-and-drainage procedure.

Dressler syndrome can cause pleural and pericardial effusions, but cardiac tamponade is an unusual result. Dressler syndrome is generally reported in patients with extensive myocardial infarctions who did not receive thrombolytic therapy.[28] In our series, 1 patient developed pericardial tamponade 2 months after anterior myocardial infarction, and the other developed tamponade 2 months after an inferior myocardial infarction. Neither of these patients received thrombolytic therapy during the acute phase of infarction. This suggests that pericardial tamponade should be kept in mind as a rare but serious late complication of myocardial infarction, especially in patients who have not received thrombolytic agents.

Although tuberculosis is rare in developed countries, it remains, in developing countries, one of the most important causes of pericardial effusion and should be investigated and excluded in each patient. In our series, we found tuberculosis in only 1 patient, by the presence of acid-resistant bacteria in the pericardial fluid specimen.

Conclusion

Findings in our series of 50 patients with large pericardial effusions and cardiac tamponade indicate that 1) the placement of an indwelling pericardial catheter, guided by 2-dimensional echocardiography, is safe and effective for initial treatment; 2) the most common causes in a multidisciplinary hospital practice are

neoplastic pericarditis and uremic pericarditis; 3) initial assessment with clinical, serologic, and radiologic investigation and careful follow-up can in most cases yield a causal diagnosis; 4) the prognosis depends chiefly upon the patient's underlying disease; and 5) a pericardial tissue biopsy is not essential in the initial evaluation of patients. We think that the combination of echocardiographically guided percutaneous pericardial puncture and fluoroscopically guided pigtail catheter placement is a safe and sufficient treatment for patients with large pericardial effusions and cardiac tamponade. For these reasons, such treatment should be considered for initial therapy, and preferred over acute surgical intervention. We also think that the taking of a pericardial tissue specimen for pathologic examination should be considered only in patients with recurrence of an effusion, the cause of which cannot be determined by other investigative means.

References

1. Shubuta R. Disease of the pericardium. In: Alexander RW, Schlant RC, Fuster V, editors. Hurst's the heart, arteries and veins. Vol 2. 9th ed. New York: McGraw-Hill; 1998. p. 2169-204.
2. Braunwald E. Pericardial disease. In: Harrison's principles of internal medicine. Favci AS, Braunwald E, Isselbacher KJ, editors. 14th ed. New York: McGraw-Hill; 1998. p. 1334-61.
3. Salem K, Mulji A, Lonn E. Echocardiographically guided pericardiocentesis - the gold standard for the management of pericardial effusions and cardiac tamponade. Can J Cardiol 1999;15:1251-5.
4. Fontanelle LJ, Cuello L, Dooley BN. Subxiphoid pericardial window. A simple and safe method for diagnosing and treating acute and chronic pericardial effusions. Thorac Cardiovasc Surg 1971;62:95-7.
5. Alcan KE, Zabetakis PM, Marino ND, Franzone AJ, Michelis MF, Bruno MS. Management of acute cardiac tamponade by subxiphoid pericardiotomy. JAMA 1982; 247: 1143-8.
6. Kopecky SL, Callahan JA, Tajik AJ, Seward JB. Percutaneous pericardial catheter drainage: report of 42 consecutive cases. Am J Cardiol 1986;58:633-5.
7. Ziskind AA, Palacios IF. Percutaneous balloon pericardiotomy for patients with pericardial effusions and tamponade. In: Textbook of interventional cardiology. Topol EJ, editor. 3rd ed. Philadelphia: WB Saunders; 1999. p. 869-77.
8. Guberman BA, Fowler NO, Engle PJ, Gueron M, Allen JM. Cardiac tamponade in medical patients. Circulation 1981;64:633-40.
9. Markiewicz W, Barovik R, Ecker S. Cardiac tamponade in medical patients: treatment and prognosis in the echocardiographic era. Am Heart J 1986;111:1138-42.
10. Wong B, Murphy J, Chang J, Hassenein K, Dunn M. The risk of pericardiocentesis. Am J Cardiol 1979;4:1110-4.
11. Fowler NO. Cardiac tamponade. A clinical or an echocardiographic diagnosis? Circulation 1993;87:1738-41.
12. Callahan JA, Seward JB, Tajik AJ, Holmes DR Jr, Smith HC, Reeder GS, Miller FA Jr. Pericardiocentesis assisted by two-dimensional echocardiography. J Thorac Cardiovasc Surg 1983;85:877-9.
13. Sagrista-Sauleda J, Merce J, Permanyer-Miralda G, Soler-Soler J. Clinical clues to the causes of large pericardial effusions. Am J Med 2000;109:95-101.
14. Colombo A, Olson HG, Egan J, Gardin JM. Etiology and prognostic implications of a large pericardial effusion in men. Clin Cardiol 1988;11:389-94.
15. Clarke DP, Cosgrove DO. Real-time ultrasound scanning in the planning and guidance of pericardiocentesis. Clin Radiol 1987;38:119-22.
16. Heierli B, Anderes U, Follath F. Diagnosis and therapy of cardiac tamponade. An analysis of 50 patients [in German]. Schweiz Med Wochenschr 1981;111:735-41.
17. Patel AK, Kosolcharoen PK, Nallasivan M, Kroncke GM, Thomsen JH. Catheter drainage of the pericardium. Practical method to maintain long-term patency. Chest 1987;92: 1018-21.
18. Larose E, Ducharme A, Mercier LA, Pelletier G, Harel F, Tardif JC. Prolonged distress and clinical deterioration before pericardial drainage in patients with cardiac tamponade. Can J Cardiol 2000;16:331-6.
19. Tsang TS, Seward JB, Barnes ME, Bailey KR, Sinak LJ, Urban LH, Hayes SN. Outcomes of primary and secondary treatment of pericardial effusion in patients with malignancy. Mayo Clin Proc 2000;75:248-53.
20. Garcia Vasquez E. Cardiac tamponade as clinical manifestation of neoplastic process: presentation of 11 cases and review of the literature [in Spanish]. An Med Interna 2000; 17:25-8.
21. Balghith M, Taylor DA, Jugdutt BI. Cardiac tamponade as the first clinical manifestation of metastatic adenocarcinoma of the lung. Can J Cardiol 2000;16:925-7.
22. Kahl LE. The spectrum of pericardial tamponade in systemic lupus erythematosus. Report of ten patients. Arthritis Rheum 1992;35:1343-9.
23. Manresa JM, Gutierrez L, Viedma P, Alfani O. Cardiac tamponade as a clinical symptom of systemic lupus erythematosus [in Spanish]. Rev Esp Cardiol 1997;50:600-2.
24. Lee IH, Yang SC, Kim TH, Jun JB, Jung SS, Bae SC, et al. Cardiac tamponade as an initial manifestation of systemic lupus erythematosus—single case report. J Korean Med Sci 1997;12:75-7.
25. Corrao S, Salli L, Arnone S, Scaglione R, Amato V, Cecala M, et al. Cardiac involvement in rheumatoid arthritis: evidence of silent heart disease. Eur Heart J 1995;16:253-6.
26. Escalante A, Kaufman RL, Quismorio FP Jr, Beardmore TD. Cardiac compression in rheumatoid pericarditis. Semin Arthritis Rheum 1990;20:148-63.
27. Harada T, Aoyagi T, Endo Y, Uno K, Takenaka K, Nakamura F, et al. Effusive constrictive pericarditis due to rheumatoid arthritis revealed by pericardiocentesis with simultaneous pressure recording—a case report. Angiology 2002;53:105-8.
28. Shahar A, Hod H, Barabash GM, Kaplinsky E, Motro M. Disappearance of a syndrome: Dressler's syndrome in the era of thrombolysis. Cardiology 1994;85:255-8.